homozygous ZZ phenotype - and MGUS might just be a casual coincidence. However, at least three hypotheses can be discussed to explain the relationship between MGUS and α_1 AT deficiency. First, the genes for immunoglobulin heavy chains have been shown to be linked to the locus for α_1 AT on chromosome 14⁶. Second, α_1 AT may have a direct effect on immunoregulation and the relation between α_1 AT deficiency and some immune disorders has already been documented 7 . It is possible that the generalized hyperresponsiveness of the immune pathway in α_1 AT deficiency could account for the appearance of MGUS through chronic antigenic stimulation. Finally, such chronic antigenic stimulation could be due to subclinical pulmonary or hepatic impairment induced by α_1 AT deficiency 8 , although our patient did not show any evidence of disease in these systems.

References

- 1 Van Den Berghe H, Vermaelen K, Mecucci C, Barbieri D, Tricot G. The 5q-anomaly. Cancer Genet Cytogenet 1985;17:189-256
- 2 Van Den Berghe H. The 5q-syndrome. Scand J Haematol 1986;36(suppl 45):78-81

- 3 Dewald GW, Kyle RA, Hicks GA, Greipp PR. The clinical significance of cytogenetic studies in 100 patients with multiple myeloma, plasma cell leukemia, or amyloidosis. *Blood* 1985; 66:380-90
- 4 Sokal G., Michaux JL, Van Den Berghe H, et al. A new haematologic syndrome with a distinct karyotype: the 5q-chromosome. Blood 1975;46:519-33
- 5 Raskind WH, Tirumali N, Jacobson R, Singer J, Fialkow PJ. Evidence for a multistep pathogenesis of a myelodysplastic syndrome. Blood 1984;63:1318-23
- 6 Cox DW, Markovic VD, Teshima IE. Genes for immunoglobulin heavy chains and for alpha 1-antitrypsin are localized to specific regions of chromosome 14q. Nature 1982;297:428-30
- 7 Breit SN, Wakefield D, Robinson JP, Luckhurst E, Clark P, Penny R. The role of alphal-antitrypsin deficiency in the pathogenesis of immune disorders. Clin Immunol Immunopathol 1985;35:363-80
- 8 Szczeklik A, Stachura J, Pieton R, Serwonska M. Marskosc watroby I dysimmunologlobulinemia U chorego Z niedoborem alpha 1 antytrypsyny o fenotype Pi M. Pol Arch Med Wewn 1977:57:533-7

(Accepted 18 June 1990)

Subtotal colectomy following marathon running in a female patient

A C Beaumont FFARCS J P Teare MRCP
The Intensive Care Unit, Kent and Canterbury
Hospital, Canterbury, Kent CT1 3NG

Keywords: bloody diarrhoea; ischaemic colitis; subtotal colectomy

Gastrointestinal bleeding is a well documented sequelae of running^{1,2}. Occult bleeding has been reported in 8-87% of participants in long distance running³⁻⁵. Bloody diarrhoea is a rare event⁶ and is probably due to ischaemic colitis secondary to alterations in regional blood flow⁷. We report a female marathon runner with bloody diarrhoea, secondary to ischaemic colitis.

Case report

A 42-year-old woman was admitted with a 10-day history of bloody diarrhoea, vomiting, abdominal swelling and tenderness. Two days prior to admission she developed generalized abdominal pain and abdominal distension, blood in her urine, blurred vision, speech difficulties, and her menstrual period was early and heavier than normal. She was not on the contraceptive pill. Her previous history was unremarkable.

She was a keen long-distance runner, but had not trained as fully as in the previous year. Three days prior to the onset of her symptoms she had run the Canterbury half-marathon in a personal best time, and had done so on a very hot day without any refreshment during the race.

On examination she was unwell, dehydrated, afebrile, with a pulse of 92 of poor volume. Blood pressure was 160/90. Her chest was clear. She had a distended abdomen with generalized tenderness. Liver, spleen and kidneys were not palpated but ascites was demonstrated. Rectal examination revealed fresh blood. Neurological examination demonstrated cerebellar dysfunction.

Investigations: sodium 118 mmol/l, potassium 5.3 mmol/l, urea 60.2 mmol/l, creatinine 479 µmol/l, bilirubin 29 µmol/l, albumin 26 g/l, haemoglobin 13.3 g/dl, white cell count 32.4×10°/l, platelets 96×10°/l, erythrocyte sedimentation rate 16 mm/h, clotting screen: prothrombin time 17/14, thrombin time 34/35, fibrinogen degradation products >4000 µg/ml. Ascitic tap: Heavily blood stained fluid with protein content of 26 g/l. Sigmoidoscopy: Normal to 10 cm. CT brain scan: normal.

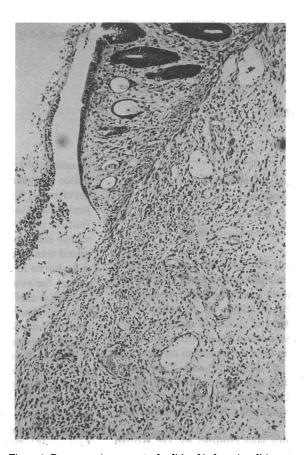


Figure 1. Demonstrating segmental colitis of ischaemic colitis type, with a focal inflammatory process extending into the muscle area with micro-abscess formation on the deep muscle aspect adjacent to the serosal fat

Treatment was started with antibiotics, dopamine, frusemide and clotting factors. Two days following her admission she underwent a laparotomy. The findings were of copious blood-stained ascitic fluid with an inflamed transverse and descending colon. A sub-total colectomy was performed. Histology demonstrated ischaemic segmental colitis (Figure 1).

Postoperatively her conscious level deteriorated and she developed grand mal seizures following which she was ventilated and haemofiltration instituted. She was eventually discharged with normal renal and neurological function. 0141-0768/91/ 070439-02/\$02.00/0 © 1991 The Royal Society of Medicine

Discussion

Previous reports of patients with confirmed ischaemic colitis secondary to marathon running have often had additional factors that have contributed to the disease. One patient who required no treatment⁷ had been taking an oral contraceptive (Ovanon) which has been linked to the spontaneous development of gut ischaemia^{8,9}. A second patient required only fluid resuscitation¹⁰, but was taking aspirin which may be involved in the prostaglandin cascade thought to be involved in vasoactive reflexes associated with regional colonic blood flow.

It is well reported that colonic blood flow may decrease up to 80% during prolonged severe exercise^{7,11} and that the additional stress of hyperthermia, and hypovolaemia would predispose to gut ischaemia. Training is said to diminish the incidence of symptoms of gut ischaemia^{6,11}.

We assume that in our patient the ischaemic colitis was due to a combination of lack of training, a very hot day¹² and lack of fluids during the race, all of which may predispose to the alteration of blood flow in the colon and ischaemic necrosis.

The cause of renal failure was not confirmed although possibly due to a combination of dehydration from her toxic clinical state and the disseminated intravascular coagulation. She had no evidence of rhabdomyolysis.

The cerebellar signs remain unexplained, but may be related to her disseminated intravascular coagulation, hyponatraemia or uraemia.

References

- 1 Cantwell JD. Gastrointestinal disorders in runners. JAMA 1981:246:1404-5
- 2 Fisher RL, McMahon LF, Ryan MJ, Larson D, Brand M. Gastrointestinal bleeding in competitive runners. Dig Dis Sci 1986;31:1226-8
- 3 Porter AMW. Do some marathon runners bleed into the gut? BMJ 1983;287:1427
- 4 Stewart JG, Ahlquist DA, Mcgill DB, Ilstrup DM, Schwartz S, Owen RA. Gastrointestinal blood loss and anaemia in runners. Ann Intern Med 1984:100:843-5
- 5 McMahon LF, Ryan MJ, Larson D, Fisher RL. Occult gastrointestinal blood loss in marathon runners. Ann Intern Med 1984:100:846-7
- 6 Fogoros RN. Runner's Trots. Gastrointestinal disturbances in runners. JAMA 1980:243:1743-4
- 7 Heer M, Repond F, Hany A, Sulser H, Kehl O, Jäger K. Acute ischaemic colitis in a female long distance runner. Gut 1987;28:896-9
- 8 Hurwitz RL, Martin AJ, Grossman BE, Waddell WR. Oral contraceptives and gastrointestinal disorders. Ann Surg 1970;172:892-6
- 9 Cotton PB, Thomas ML. Ischaemic colitis and the contraceptive pill. BMJ 1971;3:27-8
- Moses FM, Brewer TG, Peura DA. Running-associated proximal haemorrhagic colitis. Ann Intern Med 1988;108:385-6
- 11 Clausen JP. Effect of physical training on cardiovascular adjustments to exercise in man. Physiol Rev 1977;57:779-815
- 12 Costill DC. Physiology of marathon running. JAMA 1972; 221:1024-9

(Accepted 3 July 1990. Correspondence to Dr P Tearne)

Meeting reports

Forensic and legal aspects of accident and emergency medicine

Keywords: accident; co-operation; medical witness

The Section of Clinical Forensic Medicine has been happy to welcome to our meetings members of other sections where there is an interest in legal medicine. It was therefore a natural extension of this policy which resulted in The Section of Accident & Emergency Medicine inviting members of the Forensic Medicine Section to join them in their symposium on Forensic and Legal Aspects of Accident and Emergency Medicine held at the Royal Society of Medicine on the 25 January 1991, and this report is published with the agreement of the President Mr M Tabone-Vassallo.

The morning session was chaired by Dr Robin Moffat, President of the Section of Clinical Forensic Medicine, and he introduced the first speaker, Dr David Jenkins, former President of the Association of Police Surgeons, Forensic Medical Examiner to the Metropolitan Police and member of Council of the Section of Clinical Forensic Medicine. He addressed the meeting on the subject of the problems of assessing injuries in police custody. The welfare and safety of the prisoner in the police station is the responsibility of the police, and they depend upon the forensic medical examiner to assess the severity of the injury or illness, the probable cause, to give the appropriate treatment and to decide whether the prisoner is fit to be detained, or whether he should be admitted to hospital. He depends upon the co-operation of the casualty officer and Dr Jenkins stressed the importance of this, pointing out that the police surgeon is always happy to offer his or her services to the young and possibly inexperienced junior hospital doctor. The need for a thorough examination of the prisoner, the taking of photographs where indicated and meticulous notes of all injuries found was emphasized. It was pointed out that it had to be borne in mind that photography by the medical officer for evidential purposes is not permitted, but it may be useful in recalling the scene at a later date. The examiner is aware that once his examination is complete there is no medical supervision whatsoever and he must ensure that the police officer in charge is adequately instructed as to management. In addition to treating injuries, the examiner will be considering whether the findings support a possible charge of attempted murder or serious assault. He or she must interpret the injuries and must differentiate between injuries caused by assault and those which may have been self-inflicted or caused by accident. In the discussion which followed, it was clear that the forensic medical officer does undertake minor surgical procedures such as suturing, and there was much debate about the management of the injured prisoner who is under the influence of alcohol or drugs, and it was agreed that the provision of suitable facilities for dealing with this often difficult problem is a matter of priority.

Just as the relationship between accident and emergency and the forensic medical examiner is of great importance, equally so is that between the Department of Emergency Medicine and the police and this was the subject of the paper by **Dr John Gosnold** (Consultant in Accident and Emergency Medicine at Hull Royal Infirmary). Dr Gosnold stressed

Report of
joint meeting
of Sections of
Accident &
Emergency
Medicine and
Clinical Forensic
Medicine,
25 January 1991

0141-0768/91/ 070440-04/\$02.00/0 © 1991 The Royal Society of Medicine